cavity, the surgeon injured the sensory conducting fibres going to that locality, but it would be hard to understand how only those for the fingers and the platysma could have been injured.

The exacerbation of all the symptoms on physical exertion and emotion is easily explained in this way: The tumor amounted practically to a blood-cyst, which communicated with the arterial system. Any increase in the activity of the heart (exertion and emotion) necessarily increased the supply of blood to the cyst, increasing the pressure and stimulating the ganglionic cells.

A later history of this case will be published in due time.

RESPIRATORY PARALYSIS AFTER DIPHTHERIA AS A CAUSE OF PULMONARY COMPLICATIONS, WITH SUGGESTIONS AS TO TREATMENT.

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A PERUSAL of some of the literature of diphtheritic paralysis leads me to think that too little attention has been paid to the effects of paralysis of the muscles of respiration on the movements of the chest and to the affections of the thoracic viscera which may result therefrom.

That the muscles of ordinary respiration are liable to this form of paralysis is presumably accepted by every one; yet I have been surprised to find how comparatively few observers have recognized this complication, even in hospital practice, where it should be relatively frequent. Moreover, the condition is not absolutely rare, seeing that in the course of the last four years at least fifteen cases of it have come under my personal observation.

Recent writers on diphtheria, whilst discussing at length the varieties of cardiac paralysis, say little or nothing about the effect of diaphragmatic or intercostal paralysis on the lungs, and through them on the pulmonary circulation and the heart.

This paper is the outcome of observations made on fifteen cases of diphtheritic paralysis in which the muscles of ordinary respiration, especially the diaphragm, became more or less involved. Thirty-four consecutive cases of the disease, treated in the wards of the Northeastern Children's Hospital, furnish thirteen of these. One case, which occurred in 1886, first drew my attention to the subject; the others occurred more recently. They are distributed as follows: One case in 1886, five in 1888, and nine in 1889. I have met with only one doubtful case during the present year, although several patients with diphtheritic paralysis have been under treatment in the hospital.

These figures obviously afford no index of the relative, much less of the absolute, frequency of the complication; but they undoubtedly show that it is not altogether rare.

It is generally met with in "bad" cases—that is, in those in which the paralysis is widely distributed and profound; and this circumstance probably accounts for its being sometimes overlooked, as it is not usually deemed expedient to submit such cases to very searching examination, and the signs of paralysis of the diaphragm and of other respiratory muscles are often but slight deviations from the healthy movements, such as might easily be missed in the presence of more striking or urgent paralysis.

The classical signs of diaphragmatic paralysis are: reversal of the respiratory movements of the epigastrium and hypochondria; dyspnœa on exertion or excitement; diminished force of cough, sneezing, spitting, etc.; and loss of compressive action of the abdominal muscles, difficulty in defecation, etc. (von Ziemssen). The more constant and obtrusive physical signs presented by the cases now under consideration were:

1. Increased movement of the lower ribs, observed in ten out of fourteen cases; not noted in three.

2. Altered movements at the epigastrium during respiration in twelve out of fourteen cases.

3. Altered character of cough and voice. Noted in ten cases, but probably present in all.

These signs are well illustrated by the following cases:

CASE I.—Henry H., æt. four years, admitted to the Northeastern Children's Hospital on August 18, 1888.

History: Has never been robust. In the middle of July he suffered from a painful and severe affection of the throat, with enlargement of tonsils and discharge of blood-stained mucus from the mouth. There has been some ulceration of the mouth until a week ago. On August 17th a nasal twang was first noticed, and there was regurgitation of food through the nose. On August 20th he was taken into the garden, but fell over in attempting to pick up something, and complained of his right leg, which was powerless. The right arm became weak on August 22d, and the left leg on the 28th. The parents stated that the house-drains had been out of order for some time, and that two cases of diphtheria had occurred four months before in a neighboring house.

State on admission: Markedly anæmic. Occipital glands enlarged. Great general weakness. Temperature 98.6° F. Pulse 120, irregular and intermittent. Heart in normal position; intermittent action; no murmurs. Respiration normal (*sic*), 16 per minute. Tongue covered with a patchy white fur. Liver and spleen not enlarged. Some scybala felt in hypogastric and left iliac regions. There is general muscular weakness. The soft palate is motionless. The pupils are equal and active.

The superficial reflexes are all present, but the knee-jerks are absent. Some urine, which had to be drawn off by catheter, was normal.

August 30. Heart very irregular and tumultuous last night. (Ordered ext. convallariæ, gr. ½ every two hours, and brandy at the rate of 3 ounces in the twenty-four hours.) The following note was dictated at my visit that afternoon: "There is marked pallor of the integuments and a somewhat earthy tint. Breathing is deep, rather slow, and entirely thoracic. The epigastrium falls in during the deepest inspiration, and no downward movement of the abdominal viscera can be detected. The depression at the epigastrium fills up again during expiration. Diaphragmatic paralysis is apparently complete. All the intercostal muscles seem to act strongly. The heart's action is regular and rapid, with sharp sounds." Is taking liquid food and brandy in sufficient quantity. (Ordered a hypodermic injection of brandy and ether every three hours.)

31st. Very restless last night. Breathing labored, alæ nasi acting. Lips and ears dusky. Pulse very small; too rapid to count. Urine contains much albumin and some pale urates. "The epigastric sinking is not quite so obvious to-day, but the abdominal wall is occasionally seen to bulge distinctly during expiration. The expansion of the lower ribs is also rather less than yesterday, and there is a very slight sinking in of the lower intercostal spaces during inspiration. Air enters well into the upper part of the lungs, but very imperfectly along the anterior margins and at the extreme bases. There are no râles. The temperature remains down. Pulse 204."

September 3. Patient is looking rather better, although the temperature has been somewhat raised during the last three days. Pulse 138, full, of good tension, and regular. Breathing still costal, with inspiratory recession of epigastrium. Respiration 36. Air enters well (*sic*). The loss of power in the arms has increased since admission; the child cannot now raise his hands to his head. The grasp is very weak on both sides. The plantar and epigastric reflexes cannot be obtained. The voice is still nasal and indistinct, and the child appears unable to cough. There is constipation since four days. (To have stimulant hypodermics every six hours.)

4th. Evening temperature yesterday 100.6°, this morning 99.6°. Yesterday, at 4 P. M., the patient suddenly turned very pale, and the breathing became more rapid (42) and labored, with irregular pulse (144). At the same time the inspiratory recession at the epigastrium became more marked. He was given a stimulant hypodermic, and the daily allowance of brandy was increased to four ounces. Breathing is quieter this morning, but there is still some cardiac irregularity. Tongue is dry and furred.

6th. Evening temperature yesterday 101°. Heart's action still irregular and excited; pulse-beats do not correspond to the contractions of the heart. Epigastric recession about the same. Toward evening the temperature rose to 103°, and signs of consolidation, with moist râles, were detected at the right base. The child was now very restless, breathing about 60 per minute, with a pulse of about 170. There was gradually increasing asphyxia during the next few hours, against which free stimulation and artificial respiration availed nothing, and death closed the scene on September 6th.

Necropsy: On opening the chest the heart is uncovered by lung. The diaphragm rises to the level of the third interspace. There are no pleural adhesions. *Right lung:* The whole of the lower lobe is of a dark-blue color, and entirely devoid of air. The lobules are well seen on the surface. The cut section shows numerous slightly raised patches of broncho-pneumonia, of a lighter color, grouped around the smaller bronchi. The lung tissue is soft and unduly wet. The posterior part of the right upper lobe shows recent pneumonia, contains but little air, and presents a granular section. The rest of the lung is œdematous and crepitant. The *left* lung presents exactly similar changes throughout the lower lobe, though in a somewhat less degree. The upper lobe is œdematous. The heart is firm, but pale, the pericardium healthy. The abdominal viscera appear healthy to the naked eye.

CASE II.—Willie W., æt four years. Admitted October 24, 1889. Five weeks ago he had a bad sore-throat, and was brought to the hospital a week ago because he could not walk.

State on admission: A fair, well-nourished boy; rather fretful. Is unable to stand without support. No ocular paralysis. Voice natural. No trouble in swallowing. Movements of palate rather sluggish. Movements in respiration normal; no evidence of failure of diaphragm. Heart and lungs healthy. Knee-jerks absent; superficial reflexes present.

October 29. There was some difficulty in swallowing liquids yesterday for the first time, and to-day the patient coughs and splutters with each attempt. Palate is quite motionless.

November 2. Some improvement in swallowing. Pulse 96, of good quality. Temperature subnormal.

6*th.* There is a faint internal squint in the right eye, and the lateral movements of the eyeballs are limited. Voice distinctly nasal since two or three days. The child very rarely speaks. Is still able to swallow slowly and with much care. The movements of the lower ribs are somewhat excessive, and the abdomen only moves slightly during respiration. The epigastrium, however, still bulges occasionally during inspiration.

8*th.* To-day there is a slight but undoubted bulging of the epigastrium during expiration. There is no duskiness of face or lips. Cough is rather troublesome, especially after swallowing, but still retains its explosive character.

9*th.* The abnormal movements are rather more accentuated to-day, and the cough is decidedly less forcible. Pulse 88, of fair strength, but rather irregular. A few rhonchi are heard over the lungs. Air enters freely into every part. Was ordered to be fed by nasal tube, and to have artificial respiration performed for ten minutes three times a day.

11*th.* No obvious change in patient's condition. The expiratory bulging of the abdomen is a little more marked, and there is distinct recession of the abdominal wall during inspiration.

12*th.* General condition rather improved. The movements of the lower ribs are very obvious, and distinctly excessive, when compared with those of a child in an adjacent cot. Expansion is more obvious than elevation.

14*th.* Last night the child became restless and paler, and the breathing slower. This morning he is quieter, and the lips have regained a better color. He can now scarcely move his head from side to side. Yesterday the arms became weak, and their movements incoördinate. Breathing is still entirely thoracic. Respirations 24 per minute, long and sighing. No physical signs at the bases. Pulse 124, full, but very compressible. (Artificial respiration for ten minutes every six hours.)

15*th*. At 11 A. M. the child suddenly vomited, the face became dusky, respiration came in gasps, and the pulse became extremely weak and running. A hypodermic of ether was immediately given, and artificial respiration performed. The color improved a little, but the patient did not properly rally. He vomited again at 2 P. M. Faint bronchial breathing was now made out over the bases, but soon became obscured by copious moist râles. At 4 P. M. he was slightly livid, and sweating profusely. There was gasping respiration, with very excessive movement of the lower ribs. During inspiration the epigastrium was sucked in under the ribs, and flapped outward during expiration. There was also slight inspiratory recession of the lower interspaces. Death occurred at 4.15 P. M. The temperature never exceeded 99°.

A post-mortem was not allowed.

CASE III.—William B., æt. four and a half years. Admitted September 11, 1888.

History: Had diphtheria six weeks ago. Three weeks after the onset there was regurgitation of fluids through the nose, and during the last four days he has been weak on his legs.

State on admission: Pale, well-nourished. There is general muscular weakness, with tottering, uncertain gait. Both hand grasps are feeble. The voice is nasal. The soft palate does not move during phonation, and flaps backward and forward during respiration. There is no ocular paralysis. The knee-jerks are absent, the superficial reflexes present. The heart's action is regular, the pulse good. The respiratory movements are those of health.

September 14. Voice more nasal and weaker. Pulse 120, slightly irregular. Temperature normal. Breathing 18, sighing in character. Swallowing is effected with more difficulty, and occasions attacks of coughing. The movements of the lower ribs appear to be increased in both directions. The inspiratory filling in of the epigastrium is not so marked as on admission, and the descent of the diaphragm feels less forcible. (To have artificial respiration for fifteen minutes once each day.)

17*th*. There is slight percussion dulness over both pulmonary bases, with weak breath-sounds and occasional large moist râles. Beyond this the patient's condition is not appreciably changed. Pulse 120, of fair tension, and a trifle irregular at intervals.

24*th*. The movements of the lower ribs are much increased. No downward movement of viscera during inspiration can be made out at the epigastrium. The diaphragm appears to be deeply involved. Cough is more troublesome, especially after drinking. At the right base there is deficient resonance, with very weak breath-sounds and bronchial expiration. Vocal resonance is not appreciably modified. On deep breathing a few moist râles are audible. Air enters the left base freely. Pulse 114, of good quality. Temperature varies between 98° and 99.2°. (Artificial respiration to be performed twice a day for fifteen minutes.)

25*th*. The epigastrium does not move during respiration. The abdominal note is obtained as high as the sixth rib in the right nipple line, the lung note being recognized as low as the fourth rib. Air enters very imperfectly into this lung; breath sounds very feeble at right scapular angle. The cough has quite lost its explosive character. (To be placed on his left side, and to have artificial respiration three times a day.)

28*th*. Condition much the same. There is profound anæmia, but, on the whole, the general aspect of the child is somewhat better. To-day the breathing is essentially upper costal. The epigastrium is still motionless. The cough is a little stronger, but still lacks suddenness. Air enters the right base decidedly better, but there is still some evidence of collapse. The temperature remains down. The general muscular weakness is much the same.

29*th*. Patient speaks better to-day, especially in articulating the explosive consonants. The palate moves distinctly in phonation.

October 1. There is slight (normal) movement of the epigastrium to-day. The right base continues to clear up.

2*d*. Liver dulness from fifth to seventh spaces in nipple line. Swallows without coughing since yesterday.

5*th*. Respiratory movements of lower part of chest are gaining in amplitude, but the breathing is still mainly upper costal. Pulse 90, of good quality. There is marked general improvement.

7*th*. Liver dulness reaches to margin of ribs in the nipple line; edge felt during deep inspiration. Paralysis of limbs is passing off perceptibly.

16*th*. Is putting on weight rapidly. Diaphragm now acts normally. Artificial respiration omitted two or three days ago.

24*th*. Sits up in bed, but is still unable to stand. Knee-jerks still absent.

November 1. Discharged convalescent.

CASE IV.—Percy S., æt. two years. Admitted August 12, 1889.

History: Had diphtheria five weeks ago. Recovered in a fortnight; remained weak. Ten days ago he lost the power of walking, and during the last eight days his voice has become altered in quality.

State on admission: Fair color, well nourished. No obvious ocular paralysis. Cry distinctly nasal. Palate and uvula quite motionless. Often coughs after drinking. No regurgitation of liquids through nose. Can move his legs about, but is unable to stand. Knee-jerks absent. Superficial reflexes feeble. Urine free from albumin. Rhonchi and scattered moist râles over lungs. Heart acting normally. Temperature 98.6°.

August 14. Cough troublesome. Is fed entirely by nasal tube.

16*th*. Last night, after a severe attack of coughing, the child began to breathe hurriedly and the surface became rather dusky. On examination the diaphragm was contracting, but the movements of the lower ribs in front appeared excessive. The temperature had risen to 101.2°. The distress passed off after a subcutaneous injection of brandy. Temperature this morning 102.8°. At 7 A. M. the child was sick after being fed, and rapidly became cold and blue. The diaphragm was now found to be inactive. Artificial respiration for fifteen minutes caused a marked improvement in the child's condition, and half an hour later all signs of distress had subsided; but the abdominal wall remained entirely passive excepting a trifling fulness of the epigastrium during expiration. The movement of the lower ribs was much increased. Evening temperature 102.6°. Chest not examined.

19*th*. Child doing well. Pulse 132. Temperature has fallen to normal. Artificial respiration has been systematically performed every six hours since the 16th with good results. The chest moves well. The abdomen is still motionless. There are no signs of pulmonary collapse or of broncho-pneumonia. Feeding is still entirely by the nasal tube.

20th. Yesterday afternoon the diaphragm began to act slightly. (Artificial respiration omitted.) He still coughs and splutters on attempting to swallow liquids.

24th. The movements of the abdomen are now fairly good, but still less than they should be. There is a marked general improvement. The cough has a more natural ring about it, and the voice is clearer.

29th. Improvement continues. Cough quite natural. No signs of diaphragmatic weakness remain, and the action of the lower ribs is normal.

September 7. Child now sits up in bed, but cannot yet stand. Discharged convalescent on the 11th.

CASE V.—Alfred E., æt. two years and four months. Admitted September 18, 1889.

History: About seven weeks ago he had a yellow discharge from the nose, and at about the same time the voice acquired a nasal character and fluids regurgitated through the nose after drinking. There is no history of sore-throat.

State on admission: The child is in a very prostrate condition, and makes no attempt to move in bed. The face is pale and a trifle dusky. There is a slight horizontal nystagmus, but no ocular paralysis. There is a very slight right facial paralysis. The chest moves well in respiration, and the movements of the lower ribs are not obviously increased. The abdominal walls are blown outward during expiration, and the diaphragm appears to be quite passive. There is an occasional non-explosive cough. The knee-jerks are wanting, and the abdominal reflexes are also in abeyance. There are moist râles all over the lungs, with a tendency to bronchial breathing at the left apex; no percussion dulness. Respiration 24. Pulse 128. Temperature 101.2°.

September 23. There has been no decided change in the child's condition. The temperature has been slightly above normal on one or two occasions only. The râles are more numerous over the right side of the chest. This morning the breathing became more embarrassed, and the movements of the lower ribs distinctly exaggerated. During the day the child grew more restless as the signs of asphyxia gradually increased. Artificial respiration was of no avail. Death occurred on September 24th.

Necropsy: There are no pleural adhesions on either side. The posterior portion of the right lung is extensively collapsed, the lower lobe being affected almost in its entirety. Portions of the collapsed lung sink in water. The left lung is in a precisely similar condition. The cardiac valves are healthy. There is slight distention of the right auricle. The liver and kidneys are congested.

Want of space prevents a more detailed narrative of these cases, but I may add that the whole series conformed very closely to one type. Paralysis of the palate and pharynx was present in every case, and tube-feeding was frequently rendered necessary by the difficulty of swallowing and the leakage of food into the air-passages. Obvious paralysis of the lower limbs was the rule, and in some the arms also became affected. The knee-jerks were absent in every case. In a few the muscles of the back and neck were unmistakably involved, but in others how far the weakness observed was due to actual paralysis or was simply the expression of extreme general debility was not made out.

The children were all markedly anæmic, and a dusky or earthy tint was observable in some of the more severe cases. They lay for the most part quite still in their cots, without exhibiting any signs of pain or distress. The way in which they spared their breath was very striking. In the worst cases it was impossible to get a word from them, though they would generally express assent or dissent by a nod of the head. The breathing was usually slow, sometimes sighing in character, but not labored; but after cough or other exertion signs of dyspnœa often became apparent. A short, non-explosive, ineffectual cough was also a common feature in these patients. Wherever secondary changes occurred in the lungs, such as extensive collapse or broncho-pneumonia, or when sudden cardiac failure supervened, the symptoms of these conditions naturally modified to a considerable extent the general aspect of the case. It should be added that none of the patients had been tracheotomized.

Increased movement of the lower ribs has been one of the earliest as well as one of the physical signs most constantly associated with diaphragmatic failure. It varies within wide limits, both in amplitude and duration, becoming more obtrusive when the respiratory efforts are violent, and diminishing to a considerable extent during quiet breathing. As a rule, this overaction has been most obvious where other signs of diaphragmatic paralysis were well marked, and has invariably disappeared when the diaphragm resumed work. Its character may be modified to some extent by the presence and degree of paralysis of the abdominal and intercostal muscles, but, apart from this, it is simply an exaggeration of the healthy movements. The resulting expansion of the lower portion of the thoracic cage is all the more striking, that during the same period the epigastrium is either passive or actually receding, a state of things the exact reverse of what obtains in the dyspnœa of broncho-pneumonia and collapse, to which these cases sometimes present a superficial resemblance. The phenomenon appears to be due to a compensating over-action of the scaleni and intercostals, which is intensified by the diminution or removal of the steadying action of the diaphragm on the lower ribs. It is of especial value, because by its early appearance it gives timely warning of approaching danger.

The nature of the altered abdominal movements is so clearly illustrated by the cases quoted above, that it does not call for any lengthy notice in this place. A diminution in the extent of normal movement should always arouse suspicion, and, in my opinion, cessation of movement, short of reversal, is already an indication of paresis of the diaphragm. Valuable information may also be obtained by laying the hand lightly on the epigastrium, any marked diminution or cessation of inspiratory filling-out of this region being readily appreciable in this way, particularly if the manœuvre is repeated in routine fashion at each visit.

The cough is always modified to some extent, at first becoming simply weaker, but eventually losing altogether its explosive quality (in severe cases), so that the patient is quite unable to expel any secretion or foreign body from the bronchi. Concurrently the voice becomes progressively feebler and aphonic. Strictly speaking, it is incorrect to attribute feebleness of expiration and of the associated reflex acts, cough, spitting, and sneezing, to paralysis of the diaphragm, since in all these it is relaxed. It affects them only by causing the preceding inspiration to be incomplete. But, over and above this, it is highly probable that weakness of the muscles controlling the glottic aperture is an important factor in the causation of these phenomena. The troublesome attacks so frequently observed after drinking are also to be attributed in part to the same cause, and in part to the enfeeblement and incoördination of the muscles of deglutition.

Having drawn attention to the nature of the physical signs indicating partial or complete paralysis of the diaphragm, it behooves us to consider what, if any, changes occur concurrently in the lungs, and what evidence can be adduced in support of their dependence on the paralysis of the diaphragm.

In fourteen out of fifteen cases there was some evidence of disturbed function of the lungs. But the notes of some of them are, unfortunately, so incomplete in this direction that it is impossible to estimate, with any approach to accuracy, the full significance and relative frequency of the several signs observed.

Weak breathing over the bases was noted in five cases.

Bronchial or tubular breathing in six cases, with or without elevation of temperature.

Percussion dulness in four cases.

Moist râles over the backs in nine cases.

In other words, there was evidence in a majority of the cases of imperfect entrance of air into, and of some œdema of the hinder part of the lungs, more especially toward the bases, with condensation or consolidation of the organs in several of them. This state was, no doubt, to some extent aggravated by feebleness of circulation, and by the enforced maintenance of the dorsal decubitus for a prolonged period; but the sequence of events in many cases, quite apart from post-mortem evidence. was always strongly suggestive of a causal relationship between the lung condition and enfeeblement of the diaphragm. This is well illustrated by the cases recorded above.

In Case I, breathing was normal on admission; the heart irregular.

On the third day the diaphragm became paralyzed, and the heart more irregular and tumultuous. On the fourth day there was more dyspnœa, and air was entering imperfectly at the bases of the lungs, but no râles were audible. On the seventh day there was a fresh attack of dyspnœa again, attended by cardiac irregularity, and the signs of diaphragmatic paralysis became accentuated, whilst the temperature began to rise. On the eighth day the dyspnœa was much increased, and consolidation and œdema râles were detected at the right base. Throughout the illness the ribs were expanding freely. After death, on the tenth day, both lower lobes were found completely airless, and studded with patches of broncho-pneumonia.

This patient was not on his back more than ten days, so that decubitus can have had but little to do with the state of his lungs, and it is not easy to see in what way feebleness of the circulation could have induced wholesale pulmonary collapse. It is far more likely that the transient attacks of heart-failure were caused by the more or less sudden collapse of successive portions of the lungs, whilst the sudden aggravation of all the symptoms, with rise of temperature on the eighth day, finds a sufficient explanation in the onset of pneumonia of the right-upper lobe, and patchy pneumonia on the bases. On the other hand, it stands to reason that if the diaphragm fails to descend (contract) efficiently, there will not occur within the chest a sufficient lowering of pressure to overcome the elasticity of the lungs, and admit of their proper expansion, and it is reasonable to suppose that such an effect would be more immediately exerted on that part of the lungs in the neighborhood of which it occurred. This view affords a rational, and, I believe, a true explanation of the manner in which collapse occurs in these cases.

In Case II. the diaphragm became gradually paralyzed on the thirteenth day, but, judging by the incomplete character of the physical signs, the loss of function was only partial, and the over-action of the ribs was apparently able to compensate it. The patient improved somewhat under artificial respiration from the fourteenth to the eighteenth day, although the paralysis of the trunk muscles was still on the increase. On the nineteenth day there was a bad attack of cardiac failure, from which the patient did not rally completely. From this moment there was evidence of total paralysis of the diaphragm, but it was impossible to ascertain whether this event preceded or accompanied the heart disturbance. Shortly afterward weak bronchial breathing was made out over the bases, and rapid œdema of the lungs set in under observation. I have little doubt that in this case also a necropsy would have revealed extensive collapse and œdema of the bases.

Case III. developed diaphragmatic paralysis on the fourth day. Three days later there were signs of œdema and collapse of both bases (relative dulness, weak breathing, and moist râles, without elevation of temperature), with occasional slight cardiac irregularity. On the fifteenth day the collapse of the right base appeared to be considerable. The abdominal note reached as high as the sixth rib in the nipple line, and the lung note ceased at the fourth rib. He began to improve on the twentieth day. Air began to enter the right base more freely as soon as the diaphragm showed signs of returning activity, and the lower level of the liver dulness simultaneously descended from the sixth rib to the normal level in the course of five or six days. It will be observed that the right lung was chiefly affected, and that from the eighteenth to the twenty-third days the type of breathing was upper costal, the lower ribs gradually resuming work after that date. It is probable that in this case the right half of the diaphragm suffered more than the left, and that the effect on the subjacent lung was intensified and extended by the loss of function of the lower ribs. The ultimate recovery of this patient was, I believe, materially assisted by the systematic performance of artificial respiration.

Case V. was admitted with profound affection of the diaphragm and evidence of marked œdema of the lungs. After death, which occurred on the sixth day after admission, the bases of both lungs were found to be collapsed in almost their whole extent.

Evidence of a similar kind could be obtained from any one of the cases in the series, if it were desirable. But I think the references just made are sufficient to establish relationship between paralysis of the diaphragm and loss of function in the subjacent lung. It is not contended that collapse invariably results, but rather that *the loss of function in the lungs is, cæteris paribus, proportionate to the degree of paralysis of the diaphragm.*

I will now briefly relate two cases which indirectly confirm the view just enunciated, and tend to show that it holds good for other parts of the thorax.

Case VI.—Joseph H. was admitted under my care at the North-eastern Children's Hospital in May, 1886, with well-marked diphtheritic paralysis of the trunk and limbs. On the tenth day some irregularity and increased frequency of pulse were noted. Meanwhile the paralysis was becoming more profound, and the cough was altogether ineffectual and non-explosive. There is no detailed note as to the character of the respiratory movements, except that "the movements of the ribs appeared to grow less day by day."

On the seventeenth day respiration suddenly became embarrassed, and lips and extremities cold and bluish. Examination of the chest showed that the right side was practically immobile, with the exception of the two or three upper ribs. The percussion note was much impaired over the right front and base. At the apex in front the breathing was loud and tubular, weak over the middle lobe and almost inaudible at the base; behind, above the level of the scapular angle, the breath sounds were fairly loud and bronchial. There was no pain of any kind, and the temperature was not raised more than a degree. On the eighteenth day the child was much weaker; there were impaired resonance and weak tubular breathing, without râles, all over the right front, and exaggerated breathing over the left lung. The heart was not displaced; the backs were not examined. The movement of the right half of the chest was only just appreciable, and the epigastrium receded during each inspiration, whilst it bulged slightly during expiration, and more distinctly so during cough, which was very frequent and ineffectual. The temperature was beginning to rise (100°), the pulse 144, and more thready. During the next two days the child grew worse, and the temperature rose gradually to 102.4°. On the twenty-first day, however, the case began to look more hopeful. The change for the better was rather sudden, and amongst the first favorable indications were a return of movement in the right chest and a rapid disappearance of the signs of diaphragmatic paralysis. Examination of the backs revealed bronchial or tubular breathing over the right apex and posterior apex of the lower lobe, and distant weak breathing at the extreme base of the right side, and exaggerated breathing over the left. On the twenty-fifth day the two sides of the chest moved equally. Three days later, the breathing at the right apex had lost its tubular quality, and from that time recovery was uninterrupted.

Commenting on this case in the *Lancet* (1887), I inclined, somewhat hesitatingly, to the opinion that the lung was in a large measure collapsed, but only hinted at a possible relation to muscular paralysis. Looking back on this case now with the light of subsequent experience, I have little or no hesitation in attributing the physical signs to wholesale collapse of the right lung, induced by the immobility of the right half of the chest and the paralysis of the diaphragm.

It does not seem necessary to invoke the aid of paralysis of the pneumogastric to account for these phenomena. To begin with, it is not very clear in what way paralysis of the bronchial muscles could produce collapse of the lung, although it probably tends to promote œdema by its indirect effects on the local circulation; and, moreover, the occurrence of the physical signs under consideration in those portions of the lungs alone which are subjacent to a paralyzed chest-wall renders it highly improbable that they are dependent on causes acting within the lung itself. On the other hand, the expansion of the lungs is in large measure a purely physical process, and it seems to me that the mechanical defect in the apparatus which must occur as a result of paralysis of the chest-wall adequately explains the changes which have been found.[1]

The second case, to which my attention has been directed quite recently, is also of great interest. It was communicated to the Clinical Society of London in 1876, by the late Dr. J. Pearson Irvine, under the following suggestive title: "A Case of Diphtheritic Paralysis Simulating Extensive Lung Disease,"[1] of which the following is a brief abstract.

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[1] It is of interest to note in this connection that in a series of experiments upon animals to determine the effects of section of the phrenic nerves, Drs. Hare and Martin, of Philadelphia, inform me that "the lungs were found collapsed in those cases in which death occurred as the result of section of the phrenic nerves."

CASE VII.—Y. W., a girl, aged six years, had suffered from symptoms of bronchitis for fourteen days. A brother, aged four years, had died six weeks previously of scarlet fever, and on close inquiry it was subsequently ascertained that both he and the patient herself had had diphtheria at that time. Her illness began rather suddenly with short frequent cough and considerable dyspnœa. She was taken off her feet almost at once. She was feverish the first two or three days, but had suffered no pain.

When first seen she was dull and listless of aspect with a queer look about the eyes, and a drooping head. The temperature was normal, the respirations (14) shallow, slow, but not laborious. She had a continuous cough, as though to clear the throat. There was decided flattening of both infraclavicular regions with almost complete absence of movement. The flattening was extensive and uniform, and it appeared as though the upper part of the chest had fallen in. The bases expanded unusually fully, and there was a strong action of the diaphragm (*sic*). There was marked loss of resonance above and under both clavicles as low as the third ribs, and on each side toward the sternum it terminated at the level of the second ribs in absolute dulness, in what appeared to be an increased area of superficial cardiac dulness. In the supraspinous fossa there was what appeared to be absolute dulness, but the rest of the back was resonant, the bases, indeed, being abnormally so. Every variety of bronchitic râle could be heard. At the apices there were loud sonorous râles not concealing what seemed to be true tubular breathing. There was clear whispering pectoriloquy at both apices.

Four days later she was again brought to the hospital, having been worse in the interval. The chest signs were unchanged, the temperature remained normal. Pulse 60, respirations 14. No distress. The head drooped more, and there was decided left strabismus. The paralysis of the trunk and limbs was also more marked. The speech had become thick and nasal, and on swallowing there was distinct pharyngeal gurgling. The flattening was pointed out to the mother, who at once remarked that the child had been remarkably full chested before the present illness.

She was in much the same state when next seen three days later. A week after this there was a striking change. The head was all but erect, the squint had nearly disappeared, the walk was no longer ataxic, and the child was quite cheerful. The improvement occurred quite suddenly. There was now an increase of resonance below the clavicles, and a distinct diminution of dulness around the cardiac area. The breathing was tubular at the right apex, weak elsewhere, with a complete absence of râles. The upper regions of the thorax still moved imperfectly. A fortnight later she was to all appearance quite cured. There still remained some impairment of movement below the clavicles, but these regions had now become more prominent. The lung gradually invaded the area of increased cardiac dulness, which became reduced to normal dimensions. Tubular breathing below the right clavicle persisted for some time longer.

Commenting on this case Pearson-Irvine makes the following remarks:

"The disease was peculiar in many respects, particularly as regards the bronchial attack, which seemed to be so extensive as judged by physical signs, and yet so unlike in its general symptoms and course to that of an ordinary general catarrh of the tubes . . . All the physical phenomena met with

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[1] Clinical Transactions, vol. ix.

> at the upper parts of the thorax appear to me to have been due to a paralysis of the muscles concerned in the elevation and expansion of these parts.
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> "In consequence of this paralysis and the increased action of the diaphragm not only were the apices reduced to the expiratory condition, but even to a condition of temporary collapse, or one allowing accumulation of secretion, so that there were flattening and loss of resonance with tracheal breathing, and also retraction of the edges of the lungs, with consequent increase of the cardiac dulness. And while the symptoms of bronchitis might in part be due to this same cause, I would ask whether a paralysis of the muscles of the bronchial tubes could not better explain this condition. . . . The bronchial symptoms also disappeared suddenly, as if from a sudden recovery of the ataxic muscles, while the changes ascribed to paralysis of the external muscles continued for some time afterward."

This case appears to me strongly to support the view put forward in this paper, for in it the upper portion of the lungs was placed in very much the same circumstances as were the bases in those cases in which the diaphragm was paralyzed; and the condition of the apices, as far at least as can be judged by physical signs, bore a very close resemblance indeed to that revealed post-mortem in Cases I. and V., and which, I believe, was present in varying degree in all the others.

As I have already suggested, the state of imperfect expansion (or of partial or complete collapse) into which the subjacent lung is thrown by paralysis of the chest-wall or diaphragm, is just such a one as to favor the occurrence of local œdema, quite apart from paralysis of bronchial muscles, while the extra work put upon the right side of the heart when the collapse is extensive, is well calculated not only to aggravate the local condition, but to cause œdema to become general throughout the lungs. Such an event is all the more likely to take place in cases like these where the heart is often already weak, and not in a state to bear any additional strain.

To recapitulate, the facts brought forward are held to justify the conclusion that *paralysis of the diaphragm or other parts of the chest-wall tends to induce loss of function in the subjacent lung, which is, cæteris paribus, proportionate to the degree of paralysis, and results in more or less collapse and œdema of the pulmonary tissue.*

It may be objected that the immobility of the chest-wall which is held to be instrumental in producing collapse of the lung is, in reality, a secondary event, a result and not a cause of collapse, just in the way that the chest-wall moves relatively little over a lung which is rendered inactive by consolidation or extensive adhesions. The answer to such an objection is, that, were it true, there should have been diminution of movement, if not actual recession, of the lower ribs in Cases I., III., and V., for instance, in which the pulmonary bases were certainly either collapsed or consolidated, and not a distinct increase of movement, such as was actually observed.

It is not my intention to enter here upon the question of the general treatment of diphtheritic paralysis beyond remarking that the supervention of respiratory paralysis renders it more than ever imperative to take every possible precaution to guard against the occurrence of broncho-pneumonia. There are, however, one or two special points which are worthy of notice in this connection.

1. Allusion has already been made to the frequent and ineffectual cough, and to the fact that it is often incited by attempts to swallow. The adoption of tube-feeding has often proved of great value in allaying this distressing and exhausting symptom. It should be put into practice as soon as swallowing is followed by attacks of coughing. A soft red-rubber catheter of suitable size introduced through the nose answers the purpose admirably. The manœuvre is of the simplest, and can be performed by any intelligent nurse, and the children very soon become reconciled to the operation. In addition to relieving cough, and enabling the quantity of food to be regulated to a nicety, this plan has the further advantage of preventing the leakage of food-stuffs into the air-passages, where they act as an irritant which the patient is generally unable to expel, and also tend to favor the occurrence of collapse.

2. I should like to say a word on the systematic employment of artificial respiration in these cases. Possible efficacy of this mode of treatment was first suggested to me by the condition of the lungs in Case I. For if collapse, resulting primarily from physical inability to expand, is the morbid state of lung to be contended against, any rational treatment must have for its object to assist expansion to the utmost. Here, as elsewhere, there is unfortunately the same difficulty in estimating what proportion of improvement is to be attributed to the treatment adopted, and what to the gradual evolution of the disease toward recovery. But I am satisfied that in several cases the ultimate recovery of the patients was materially assisted by the treatment, although in others it appeared to produce no effect, and on two occasions had to be discontinued on account of the discomfort to the patients. It was tried in ten cases, of which five recovered and five died. Until a more extended trial has been made it would be premature to express a decided opinion concerning it, but I have no hesitation in recommending others to try it.

Artificial respiration (Sylvester) should be commenced on the first appearance of any indication of paralysis of the diaphragm, for it should be borne in mind that the object in view is not so much to treat symptoms of asphyxia, etc., as to prevent, if possible, the condition of lung which gives rise to them.

In hospital the treatment is carried out by the nurses three or four times in the twenty-four hours, according to the severity of the symptoms, for a period of ten or fifteen minutes. Great care is always taken to disturb the patients as little as possible, and not to uncover them more than is absolutely necessary.

In conclusion, my best thanks are due to my colleagues, Dr. F. Charlewood Turner and Dr. Armand Semple, for their kindness in allowing me to examine patients under their care, and to make use of their case-notes.

A SERIES OF THIRTY CLINICAL LAPAROTOMIES.

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I have found considerable difficulty in arranging the title of this paper, and I do not know that the heading expresses exactly its scope. The method of operating is something of an innovation, in that the laparotomies are done in the public clinic-room, before several hundred students. These cases are presented under the supposition that their care and results may be of interest, and I have gathered all the clinical cases that have come under my control during the past two winter sessions, covering a period of about eighteen months, and operated upon by me at the public clinic of Rush Medical College.

Case I.—M. M., aged twenty-two, American, school-teacher, referred by Dr. Going, family and previous history good, presented herself for treatment with a large tumor of the abdomen. The tumor was first noticed by the patient, a year previous to her coming under my charge, as a small lump in the lower portion of the abdomen on the right side. During the last four months it had increased rapidly until it filled the entire cavity. There was nothing peculiar about it in any way. It was diagnosed as a probable single large ovarian cyst, and operation advised, which was consented to, and done before the class at the College, November 3, 1888. There was nothing found after abdominal section except a large ovarian cyst; no peculiarities, no adhesions. The patient went on to complete and perfect recovery by primary union, without interference or rise of temperature. Discharged cured November 30, 1888.

Case II.—The next operation was done December 1, 1888. Mrs. P. V., aged forty-two; married fifteen years; two children; American; housewife; family and previous history good; referred by Dr. J. G Conley. The patient had been suffering for the past two years; the history was that of an ordinary ovarian cyst, nothing uncommon being determined by examination. Abdominal section and tapping of the cyst showed the contents to be of a chocolate color. It contained sixteen pounds of fluid. There were a number of adhesions to many of the surrounding organs, but these were separated without difficulty and the tumor entirely removed. No well-defined pedicle could be found, the tumor being nourished apparently by its adhesions. In this case no unfavorable symptoms arose in the course of recovery, and she was discharged cured three weeks after the operation.

Case III.—The next operation was done March 26, 1889. Mrs. H. G., aged forty-six; American; housewife; family and previous history good; married twenty-two years; three children. Patient first